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Short Report

Rift Valley fever antibody in human sera collected after an outbreak in domestic animals in Kenya

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Rift Valley fever (RVF) virus is a member of the family Bunyaviridae, genus *Phlebovirus* (BISHOP *et al.*, 1980). As first described by DAUBNEY *et al.* (1931), the disease was a highly fatal epizootic of sheep at a farm north of Lake Naivasha, Kenya. Since that time, periodic epizootics of RVF have been recorded in Kenya. They principally involved domestic sheep and cattle populations, particularly those imported into the country. Human cases have occurred in laboratory workers and people associated with the affected animals, but are comparatively rare (DAVIES, 1975; DAVIES *et al.*, 1985). In June 1989 an outbreak of RVF occurred in domestic cattle and sheep on farms bordering Lake Naivasha, less than 20 km from the site of the 1931 outbreak (LOGAN *et al.*, 1991).

Rift Valley fever was recognized when virus was isolated from a bovine foetus brought to the Veterinary Research Laboratory at Kabete on 15 June 1989 (DAVIES *et al.*, 1991). Further investigations were made at the farm where the foetus was aborted and at 2 adjacent farms. Several RVF viral isolates were made from sheep and cattle during the outbreak and a high percentage of ruminants were RVF antibody-positive in a virus serum neutralization test (DAVIES *et al.*, 1991). RVF virus was also isolated from 5 pools of *Culez zombaensis* Theobald and one pool of *Mansonia africana* Theobald mosquitoes, (LOGAN *et al.*, 1991), which had been collected at one of the affected farms.

This report describes the prevalence of RVF virus-specific immunoglobulin (Ig) G and IgM antibody in herdsmen working on the 3 affected farms. Blood samples were taken by finger stick on to filter paper (WHATMAN® chromatography paper no. 3) to fill 2 ringed areas 10 mm in diameter. The samples were allowed to dry and then stored in a container at −20°C. They were tested by an enzyme-linked immunosorbent assay (MEEGAN *et al.*, 1987). The serum was eluted from the filter paper by soaking in the test diluent, and then further diluted to 1/400. The IgG test was performed with a mouse anti-human IgG conjugate. Samples that were RVF antibody-positive for IgG were tested for RVF IgM antibody with an IgM capture technique (KSIAZEK *et al.*, 1989). Samples were considered positive if they had mean adjusted optical density values greater than 3 standard deviations above the mean of a group of RVF antibody-negative human sera.

Four samples were collected from farm 1, where the index case occurred (altitude 1920 m; 0°48'S, 36°18'E); 18 from farm 2, where much clinical disease was seen in sheep and cattle (1920 m; 0°43'S, 36°18'E); and 8 from farm 3, where an abortion in one cow was thought to have been associated with its high titre of RVF antibody (1920 m; 0°48'S, 36°24'E).

Twelve (40%) of the 30 herdsmen tested had detectable RVF IgG antibody. Five (42%) of these 12 also had RVF virus-specific IgM antibodies. At farm 1, 2 of 4 herdsmen were IgG-positive and 1 of these was IgM-positive; at farm 2, 6 of 18 were IgG-positive and 2 of these were IgM-positive; at farm 3, 4 of 8 were IgG-positive and 2 of these were IgM-positive. No clinical disease had been apparent during the outbreak that might have been RVF, nor could the herdsmen recall experiencing any illness during that period. This sample was clearly heavily biased to detect positive cases, for all men had been closely associated with diseased cattle during the epizootic.

The widespread epizootics of RVF in ruminants in Kenya over the last 50 years were not accompanied by similar epidemics of disease in the human population. Of serum samples taken from 26 herdsmen at a farm where 80–90% of cattle were affected during an earlier RVF epizootic, only 2 were positive to RVF antigen in an indirect fluorescent antibody test (F. G. Davies, unpublished data). Other cases of RVF have been reported in East Africa (METSELAAR *et al.*, 1974; SMITHBURN *et al.*, 1949; DAUBNEY *et al.*, 1931) and RVF antibody has been shown to occur at a low level in human populations (JOHNSON *et al.*, 1983). RVF viral isolates from East Africa are considered to be as pathogenic for humans as, for example, the Egyptian strain ZH501 (BATTLES & DALRYMPLE, 1988).

There has been extensive human involvement, with mortality, in RVF epizootics in other African countries, most recently Madagascar (MORVAN *et al.*, 1991), Mauritania (JOUAN *et al.*, 1989), Egypt (MEEGAN, 1979) and South Africa (VAN VELDEN *et al.*, 1977). It is possible that the low human to animal contact ratio throughout the enzootic areas may be significant. Often 1 or 2 herdsmen will manage between 400 and 1000 animals in Kenya. In Egypt and Mauritania, family groups generally have far fewer cattle, sheep or goats and live much more closely associated with them. Vector biology may be a further and important determinant.

The samples were ethically obtained from all subjects after informed oral consent to the purpose and procedure of the serology; the data were recorded anonymously using a number system. The results have been transmitted to the managers of the farms visited during this study.

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